ResearchGate

See discussions, stats, and author profiles for this publication at: https://www.researchgate.net/publication/221850201

Developmental Coordination Disorder: A Pilot Diffusion Tensor Imaging Study

Article *in* Pediatric Neurology · March 2012

DOI: 10.1016/j.pediatrneurol.2011.12.007 · Source: PubMed

CITATIONS
26

READS
121

4 authors, including:

Jill Zwicker
University of British Columbia - Vancouver
39 PUBLICATIONS **656** CITATIONS
SEE PROFILE

Cheryl Missiuna
McMaster University
146 PUBLICATIONS **2,768** CITATIONS
SEE PROFILE

Susan R Harris
University of British Columbia - Vancouver
204 PUBLICATIONS **3,661** CITATIONS
SEE PROFILE

All in-text references underlined in blue are linked to publications on ResearchGate, letting you access and read them immediately.

Available from: Cheryl Missiuna
Retrieved on: 06 August 2016

Pediatric Neurology 46 (2012) 162–167

Contents lists available at ScienceDirect

Pediatric Neurology

journal homepage: www.elsevier.com/locate/pnu

Original Article

Developmental Coordination Disorder: A Pilot Diffusion Tensor Imaging Study

Jill G. Zwicker PhD, OT(C)[a,*], Cheryl Missiuna PhD, OTReg (Ont)[b], Susan R. Harris PhD, PT[c], Lara A. Boyd PhD, PT[c]

[a] Graduate Program in Rehabilitation Sciences, Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada
[b] School of Rehabilitation Science, Faculty of Health Sciences, McMaster University, Hamilton, Ontario, Canada
[c] Department of Physical Therapy, Faculty of Medicine, University of British Columbia, Vancouver, British Columbia, Canada

ARTICLE INFORMATION

Article history:
Received 24 August 2011
Accepted 22 December 2011

ABSTRACT

Motor deficits associated with developmental coordination disorder are not attributable to macrostructural brain abnormalities, but differences in brain microstructure may exist. Using diffusion tensor imaging, we explored the integrity of motor, sensory, and cerebellar pathways in children with and without developmental coordination disorder. In seven children with the disorder and nine typically developing children (aged 8-12 years), we measured diffusivity and fractional anisotropy of the corticospinal tract, posterior thalamic radiation, and superior and middle cerebellar peduncles. Fractional anisotropy of motor and sensory tracts and diffusion parameters in cerebellar peduncles did not differ between groups. Mean diffusivity of the corticospinal tract and posterior thalamic radiation was lower in children with developmental coordination disorder compared with control children ($P < 0.04$ and $P < 0.06$, respectively). Results were driven by lower axial diffusivity, which was significantly correlated with motor impairment scores on the Movement Assessment Battery for Children-2 for both the corticospinal tract ($r = 0.56$, $P = 0.03$) and posterior thalamic radiation ($r = 0.70$, $P = 0.003$). Reduced axial diffusivity in motor and sensory tracts may be implicated in developmental coordination disorder, but replication in a larger study is needed to confirm these findings.

© 2012 Elsevier Inc. All rights reserved.

Introduction

Developmental coordination disorder affects approximately 5% of school-age children. It is a motor disorder of unknown etiology that significantly interferes with a child's ability to perform activities of daily living [1]. Children with developmental coordination disorder struggle to complete self-care and academic tasks that require fine motor coordination. They may also experience difficulty with gross motor tasks, such as catching a ball or riding a bike. Central nervous system pathology is thought to underlie the disorder, but has only recently been a source of investigation. Recent studies indicated differences in patterns of brain activation in children with developmental coordination disorder, compared with typically developing children [2-5]. Studies have been limited to functional magnetic resonance imaging. However, other neuroimaging studies of children with developmental coordination disorder are key to a better understanding of the neurobiology underlying this disorder. In the absence of evidence for brain macrostructural differences in children with developmental coordination disorder, differences may exist at the microstructural level, which can now be measured with advanced neuroimaging techniques such as diffusion tensor imaging.

This pilot study sought to explore the integrity of motor, sensory, and cerebellar pathways, using diffusion tensor imaging in children with and without developmental coordination disorder. The major voluntary motor pathway, the corticospinal tract, and the sensory pathway from the posterior thalamic radiation have been implicated in other motor disorders, such as cerebral palsy [6]. Compared with typically developing children, children with cerebral palsy exhibited lower water diffusion along the length of their axons, and this finding was significantly correlated with functional levels on the Gross Motor Function Classification System [6]. In addition to the corticospinal tract and posterior thalamic radiation, we explored cerebellar pathways, given the hypothesized role of the cerebellum in developmental coordination disorder [7]. We selected the middle cerebellar peduncle because it carries pathways associated with the initiation, planning, and timing of motor activity [8], all thought to be affected in developmental coordination disorder [9]. We also traced the superior cerebellar peduncle because it contains pathways that carry information from the cerebellum to the primary motor and premotor areas of the cortex

* Communications should be addressed to: Dr. Zwicker; Department of Pediatrics; Faculty of Medicine; University of British Columbia; L408-4480 Oak Street; Vancouver, British Columbia V6H 3V4, Canada.
E-mail address: jzwicker@cw.bc.ca

0887-8994/$ - see front matter © 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.pediatrneurol.2011.12.007

[8]. We hypothesized that children with developmental coordination disorder would demonstrate different diffusion parameters in each of these motor, sensory, and cerebellar pathways, compared with typically developing children.

Methods

Participants

Seven children with developmental coordination disorder (six boys and one girl; mean age, 10 years and 10 months; S.D., 1 year and 6 months; range, 8 years and 7 months to 12 years and 4 months) and nine typically developing control children (six boys and three girls; mean age, 10 years and 4 months; S.D., 1 year and 7 months; range, 8 years and 1 month to 12 years and 6 months) were recruited, using methods described in detail in our previous work [4,5]. Children with developmental coordination disorder were included if they met five criteria: (1) a score of ≤16th percentile on an assessment of motor impairment, i.e., the Movement Assessment Battery for Children-2 [10]; (2) a score in the range of "suspect developmental coordination disorder" or "indicative of developmental coordination disorder" on a parent-completed measure, the Developmental Coordination Disorder Questionnaire [11]; (3) a report by the parent and child during clinical interviews that motor difficulties interfered considerably with their ability to perform daily activities [12]; (4) a score of >80 on the Kaufmann Brief Intelligence Test-2 [13]; and (5) a score of <70 on Conners' Attention Deficit Hyperactivity Disorder Diagnostic Scale, to exclude clinically significant attentional difficulties [14]. Children were excluded if they had received a diagnosis of attention deficit hyperactivity disorder, or if they were suspected of manifesting another medical condition that could explain their motor problems (e.g., cerebral palsy or autism spectrum disorder). Typically developing children were also assessed with the same measures and were included if they scored within normal range.

All participants were screened to ensure they could safely undergo magnetic resonance imaging (i.e., no metallic objects in their body or a history of major psychiatric diagnosis, claustrophobia, or seizures). We obtained ethical approval from the Clinical Research Ethics Board of the University of British Columbia, and approval for recruitment from the Vancouver Coastal Health Research Institute and the Vancouver School Board. Parents consented and children assented to participation in the study and to subsequent publication of the results.

Diffusion tensor imaging

Testing was conducted at the University of British Columbia Magnetic Resonance Imaging Research Centre on a Philips Achieva 3.0 T whole-body magnetic resonance imaging scanner (Phillips Healthcare, Andover, MD), using a sensitivity encoding head coil. Children received a single, high-resolution anatomic scan (repetition time, 12.4 ms; echo time, 5.4 ms; flip angle θ, 8°; field of view, 256 mm; 170 slices; 1-mm thickness) [4,5]. Diffusion-weighted data were collected with an echo-planar imaging sequence with a single-shot readout (repetition time, 7465 ms; echo time, 75 ms; field of view, 212 × 212 mm; 60 slices; 2.2-mm slice thickness; voxel dimension, 2.2 mm^3). A scanner-specific gradient table was used. Diffusion weighting was performed across 16 independent orientations (b = 1000 seconds/mm^2), with the acquisition of an additional minimal diffusion-weighted image (b = 0).

To map the motor and sensory tracts, we performed diffusion tensor tractography with DTIStudio software (https://www.mristudio.org), using the method of fiber assignment by continuous tracking [15,16]. Tracts that were of interest included the corticospinal tract and posterior thalamic radiation because of their association with motor function in children with cerebral palsy [6]. We initiated fiber tracking of the corticospinal tract with a seeding region-of-interest in the posterior limb of the internal capsule, followed by the placement of two limiting regions-of-interest in the white matter under the precentral gyrus and in the cerebral peduncle (Fig 1A-C) [17]. We conducted fiber tracking of the sensory tract by placing a region-of-interest at the posterior thalamus and in the white matter under the postcentral gyrus (Figure 1D,E). For both tracts, we used a fractional anisotropy threshold of 0.15 and an angle threshold of 60° [6]. To measure diffusion parameters in the cerebellum, we placed a region-of-interest in the superior and middle cerebellar peduncles (Fig 1F,G). These peduncles can be traced easily and contain efferent and afferent pathways. The superior cerebellar peduncle contains pathways from the cerebellum to the primary motor and premotor areas of the cortex, whereas the middle cerebellar peduncle contains afferent pathways associated with the initiation, planning, and timing of motor activity [8]. The first author (J.G.Z.) conducted all fiber tracking and region-of-interest placement.

Two primary diffusion statistics were calculated: (1) mean diffusivity, which is the average amount of water diffusion in three orthogonal directions (λ_1, λ_2, and λ_3); and (2) fractional anisotropy, which reflects the variance of these three eigen values and represents the direction of water diffusion. To better characterize significant and near-significant differences in mean diffusivity, we also calculated axial diffusivity (λ_1, i.e., the diffusion of water parallel to the primary direction of the tracts) and radial diffusivity [$(\lambda_2 + \lambda_3)/2$, i.e., water diffusion perpendicular to the tracts]. For each diffusion parameter, the left and right sides were averaged to obtain a single value.

Data analysis

Statistical analyses were performed using PASW Statistics, release 18.0.3, September 9, 2010 (IBM, New York, NY). The means and S.D. of mean diffusivity and fractional anisotropy were calculated for each tract and region-of-interest. Analysis of variance was used to report group differences in clinical measures, such as the Movement Assessment Battery for Children-2. Because mean diffusivity and fractional anisotropy in white matter change with development [18], analysis of covariance was used, with age as a covariate, to examine group differences in diffusion parameters in the corticospinal tract, posterior thalamic radiation, and superior and middle cerebellar peduncles. Pearson correlation coefficients were calculated to explore relationships between axial diffusivity and the degree of motor impairment. Because this study was exploratory, significance was set at $P < 0.05$ for all calculations.

Results

Participants

Descriptive results are presented in Table 1. As expected, significant differences were evident between children with developmental coordination disorder and typically developing, control children on measures of motor impairment (the Movement Assessment Battery for Children-2) and of impact on everyday function (the Developmental Coordination Disorder Questionnaire). The groups did not differ significantly in terms of age or estimates of intelligence according to the Kaufman Brief Test of Intelligence-2, but children with developmental coordination disorder were reported by parents (using Conners' Attention Deficit Hyperactivity Disorder Diagnostic Scale) to exhibit more signs of inattention or hyperactivity, compared to the control group. This finding is consistent with the literature, suggesting that children with developmental coordination disorder may manifest more attentional difficulties compared to their peers [19].

Diffusion parameters

Contrary to our hypothesis, no significant differences were evident between children with and without developmental coordination disorder in fractional anisotropy of the corticospinal tract, posterior thalamic radiation, or cerebellar peduncles (Table 1). However, the mean diffusivity of the corticospinal tract was significantly lower in children with developmental coordination disorder, compared to control children ($P < 0.04$), and a trend was evident toward lower mean diffusivity in the posterior thalamic radiation in the group with developmental coordination disorder ($P < 0.06$). To better characterize this effect, we explored group differences in axial and radial diffusivity in these two tracts. As quantified in Table 1, no differences were observed in radial diffusivity, but trends were again evident toward lower axial diffusivity in both the corticospinal tract and posterior thalamic radiation in children with developmental coordination disorder, compared with typically developing children (both tracts, $P < 0.06$). No significant differences in mean diffusivity of the superior or middle cerebellar peduncles were observed between the two groups.

Correlations with motor impairment

Given the borderline significant differences in axial diffusivity of the corticospinal tract and posterior thalamic radiation, we performed an exploratory correlational analysis to determine if any relationship existed between water diffusion parallel to the tracts and the degree of motor impairment. As depicted in Figs 2 and 3, mean axial diffusivity was significantly correlated with Movement

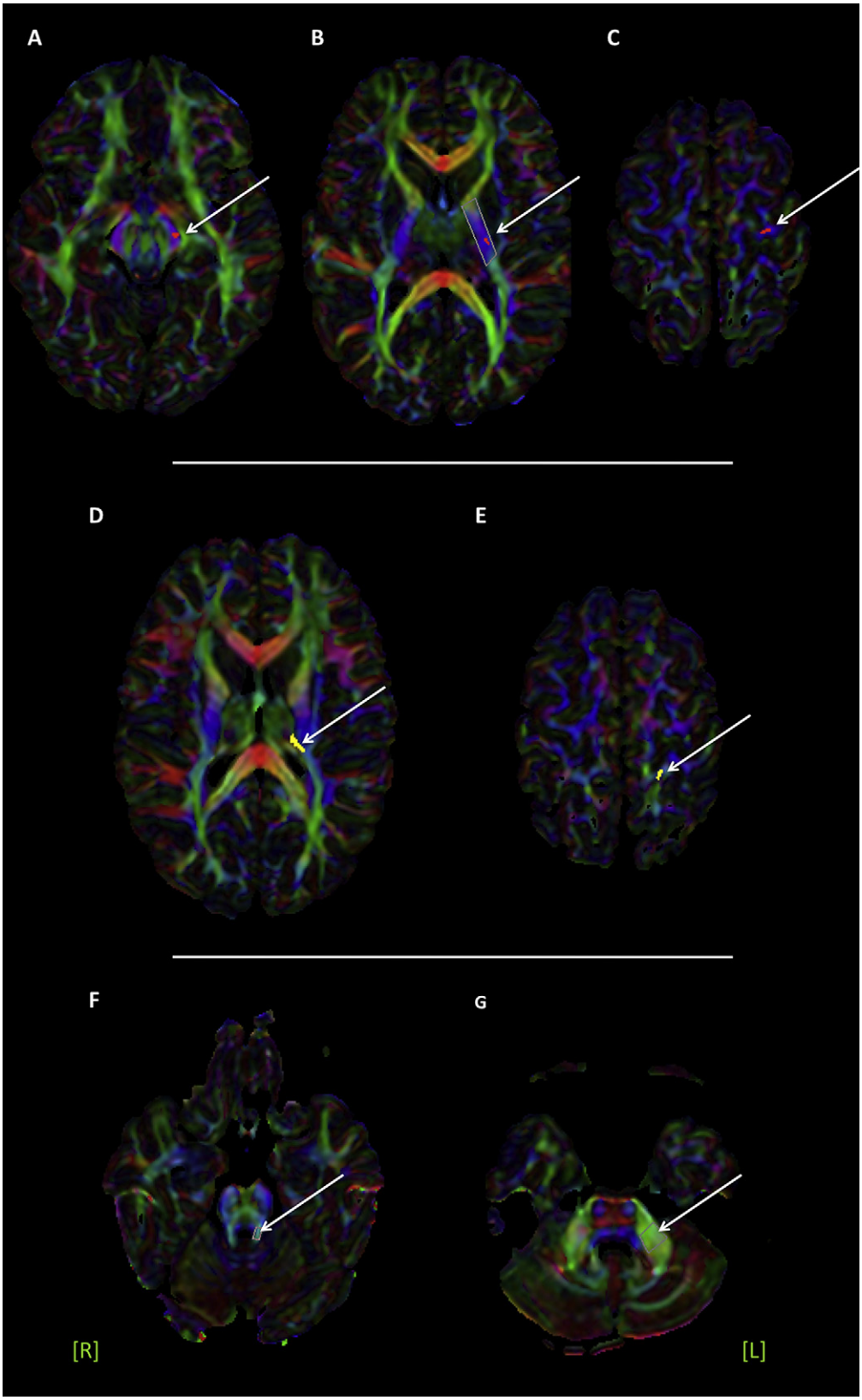

Figure 1. (A-C) Diffusion tensor images of the corticospinal tract (red). (D,E) Diffusion tensor images of the posterior thalamic radiation (yellow). (F) Diffusion tensor image of the superior cerebellar peduncle (light blue). (G) Diffusion tensor image of the middle cerebellar peduncle (green) (A) Arrow indicates limiting region-of-interest in the cerebral peduncle. (B) Arrow indicates seeding region-of-interest in the posterior limb of the internal capsule. (C) Arrow indicates limiting region-of-interest in the white matter underlying the motor cortex. (D) Arrow indicates limiting region-of-interest in posterior thalamic radiation. (E) Arrow indicates limiting region-of-interest in the white matter underlying the sensory cortex. (F) Arrow indicates region-of-interest in the superior cerebellar peduncle. (G) Arrow indicates region-of-interest in the middle cerebellar peduncle.

Table 1. Clinical characteristics and diffusion parameters according to group

	DCD		Typical		*P*
	Mean	S.D.	Mean	S.D.	
Age	10 years and 10 months	1 year and 6 months	10 years and 4 months	1 year and 7 months	0.55
MABC-2 (percentiles)	6.9	6.9	51.4	18.2	<0.001
DCDQ	36.0	10.0	65.6	8.0	<0.001
KBIT-2	112.6	11.4	110.4	15.3	0.76
CADS	57.6	6.9	48.4	6.0	0.01
Corticospinal tract					
Mean fractional anisotropy	0.62	0.013	0.63	0.028	0.83
Mean diffusivity	7.29×10^{-4}	1.47×10^{-5}	7.47×10^{-4}	1.84×10^{-5}	0.04
Mean axial diffusivity	1.34×10^{-3}	3.63×10^{-5}	1.38×10^{-3}	3.54×10^{-5}	0.06
Mean radial diffusivity	4.23×10^{-4}	1.12×10^{-5}	4.27×10^{-4}	2.89×10^{-5}	0.49
Posterior thalamic radiation					
Mean fractional anisotropy	0.48	0.024	0.48	0.023	0.84
Mean diffusivity	7.28×10^{-4}	1.60×10^{-5}	7.50×10^{-4}	2.31×10^{-5}	0.07
Mean axial diffusivity	1.15×10^{-3}	3.19×10^{-5}	1.19×10^{-3}	4.19×10^{-5}	0.06
Mean radial diffusivity	5.18×10^{-4}	1.71×10^{-5}	5.29×10^{-4}	2.48×10^{-5}	0.53
Superior cerebellar peduncle					
Mean fractional anisotropy	0.65	0.056	0.68	0.031	0.16
Mean diffusivity	3.02×10^{-3}	2.94×10^{-4}	2.40×10^{-3}	6.67×10^{-4}	0.11
Middle cerebellar peduncle					
Mean fractional anisotropy	0.72	0.025	0.73	0.029	0.43
Mean diffusivity	7.25×10^{-4}	4.27×10^{-5}	7.16×10^{-4}	2.35×10^{-5}	0.66

Abbreviations:
CADS = Conners' Attention Deficit Hyperactivity Disorder Diagnostic Scale
DCD = Developmental Coordination Disorder
DCDQ = Developmental Coordination Disorder Questionnaire
KBIT-2 = Kaufmann Brief Intelligence Test-2
MABC-2 = Movement Assessment Battery for Children-2

Assessment Battery for Children-2 scores for both the corticospinal tract ($r = 0.56$, $P = 0.03$; Fig 2) and posterior thalamic radiation ($r = 0.70$, $P = 0.003$; Fig 3).

Discussion

We used diffusion tensor imaging to compare the integrity of motor, sensory, and cerebellar pathways in children with and without developmental coordination disorder. Diffusion tensor imaging allowed for measurement of the magnitude and direction of water diffusion in brain regions, providing an indication of microstructural integrity. We determined that the mean diffusivity of the corticospinal tract, the major pathway for voluntary motor movement, was significantly lower in children with developmental coordination disorder. This finding suggests that the magnitude of water diffusion in the corticospinal tract is lower in children with developmental coordination disorder compared to control children. Although the results were not statistically significant, mean diffusivity was lower in the posterior thalamic radiation in children with developmental coordination disorder compared with control children, suggesting that the integrity of the sensory tract may also be affected in developmental coordination disorder. We did not observe any difference between groups regarding the fractional anisotropy of either tract, which suggests that the direction of water diffusion along the length of the tract is similar between the two groups of children.

The pattern of diffusion differences was somewhat surprising. Lower mean diffusivity is usually evident with increasing age [18], but this fact is unlikely to explain our findings. The group with developmental coordination disorder was slightly older than the control group, but the difference in age was not significant, and we controlled for age in our analysis. Age-related changes in white matter are typically reflected by higher fractional anisotropy (driven by changes in radial diffusivity) [18], neither of which differed between the children with developmental coordination disorder and typically developing control subjects in our study.

Lower mean diffusivity in developmental coordination disorder is also contrary to findings in other pediatric populations with neurologic impairment. Compared to control children, children with cerebral palsy [20] and those with traumatic brain injury [21] exhibited lower fractional anisotropy and increased mean diffusivity. These contrasting results may be attributable to the different mechanisms underlying these disorders, compared with developmental coordination disorder. Both cerebral palsy and traumatic brain injury have been clearly linked to macrostructural injury of the brain, but to date, overt brain injury has not been reported in developmental coordination disorder.

Recognizing that lower mean diffusivity may be a spurious finding, given our small sample size, we looked at differences in axial and radial diffusivity to better characterize our data. Our results suggest that the lower diffusivity of the motor and sensory pathways in the group with developmental coordination disorder was driven by lower axial diffusivity. Altered axial diffusivity in children with developmental coordination disorder implies that

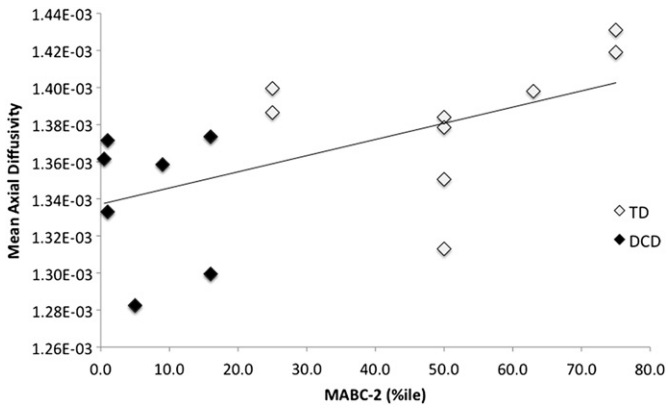

Figure 2. Axial diffusivity in the corticospinal tract according to Movement Assessment Battery for Children-2 (MABC-2) scores ($r = 0.56$, $P = 0.03$). DCD, developmental coordination disorder; TD, typically developing.

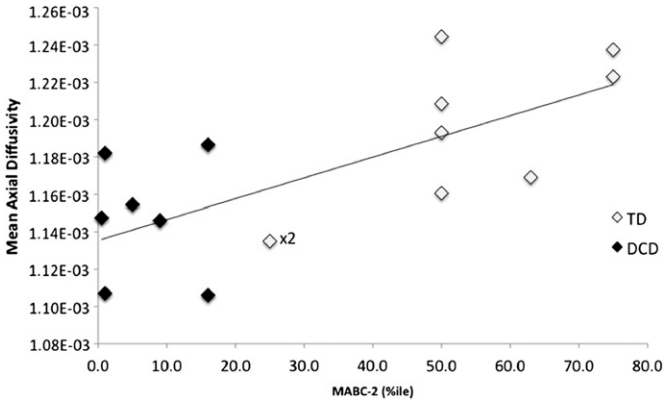

Figure 3. Axial diffusivity in the posterior thalamic radiation according to Movement Assessment Battery for Children-2 (MABC-2) scores ($r = 0.70$, $P = 0.003$). DCD, developmental coordination disorder; TD, typically developing.

differences may exist in the intrinsic characteristics of axons or in the extra-axonal/extracellular space [22], or may be attributable to reduced water content [23]. Yet the similar radial diffusivity in children with and without developmental coordination disorder suggests that no difference exists in myelination or glial cell morphology [24]. Interestingly, the same finding of reduced axial diffusivity compared with control subjects was observed in the white matter pathways of children with autism [25] and developmental dyslexia [26]. In contrast to our results, lower axial diffusivity was accompanied by reductions in fractional anisotropy in these studies [25,26]. Although we may have not been able to detect differences in fractional anisotropy because of our small sample size, changes in diffusivity without an accompanying change in fractional anisotropy are possible. In addition, lower axial diffusivity appears to have clinical significance. We observed that the axial diffusivity of motor and sensory pathways was significantly associated with scores on the Movement Assessment Battery for Children-2. Low scores on this measure were highly related to low axial diffusivity of the corticospinal tract, and even more strongly related to low diffusivity of the posterior thalamic radiation. Our results, in conjunction with the reported lower axial diffusivity in autism [24] and developmental dyslexia [25], suggest that reduced axial diffusivity may play a role in developmental disorders, perhaps by means of altered axonal microstructure [22] or reduced water content [23].

Given the hypothesized role of the cerebellum in developmental coordination disorder [7], we were surprised to observe no differences between children with and without developmental coordination disorder in mean diffusivity and fractional anisotropy in the superior and middle cerebellar peduncles. These findings imply that no microstructural differences may exist between children with developmental coordination disorder and typically developing children along the efferent and afferent pathways contained within these cerebellar peduncles. These results, combined with the relative underactivation of the cerebellum in children with developmental coordination disorder (as indicated by functional magnetic resonance imaging) [5], suggest that the hypothesized cerebellar involvement in the disorder may be related to the cerebellum itself, rather than to the connections to and from it. Examining cerebellar volumes and other macrostructural differences between children with and without developmental coordination disorder would be worth investigating, to test this hypothesis.

The main limitation of our study involves the small sample size. However, this pilot study was performed because no other work, to the best of our knowledge, has examined the integrity of motor, sensory, and cerebellar pathways in children with and without developmental coordination disorder. Our work can be used to calculate sample sizes for larger diffusion tensor imaging studies of children with developmental coordination disorder. In addition to larger sample sizes, future studies could expand on this work by incorporating more diffusion directions in the diffusion tensor protocol to increase the accuracy of diffusion measures. Our study may have been strengthened by using the stricter cutoff of <5th percentile on the Movement Assessment Battery for Children-2, although we still observed significant differences using a more liberal cutoff score. Given our preliminary results, it would be interesting to determine if any differences exist in the diffusion parameters of motor and sensory pathways in children who score in the lower ranges of the Movement Assessment Battery for Children-2 (1st to 5th percentiles), compared with those manifesting borderline motor impairment scores (6th to 15th percentiles).

Conclusion

The results of this pilot study indicate that the mean diffusivity of motor and sensory pathways is lower in children with developmental coordination disorder, compared to control subjects. The axial diffusivity of the corticospinal tract and posterior thalamic radiation is highly and significantly correlated with the degree of motor impairment, as measured by the Movement Assessment Battery for Children-2. These data suggest that differences in the intrinsic characteristics of axons or in the extra-axonal/extracellular space may underpin some of the deficits observed in children with developmental coordination disorder. To verify this hypothesis, our results should be replicated with a larger sample.

This study was funded by a grant from the Vancouver Coastal Health Research Institute and Foundation to L.A.B. At the time of this study, J.G.Z. was funded by a Quality of Life Strategic Training Fellowship in Rehabilitation Research from the Canadian Institutes of Health Research Musculoskeletal and Arthritis Institute and a Senior Graduate Training Scholarship from the Michael Smith Foundation for Health Research. J.G.Z. is currently supported by the Child and Family Research Institute (Vancouver, British Columbia), the Canadian Child Health Clinician Scientist Program, the Michael Smith Foundation for Health Research, and NeuroDevNet. At the time of this study, C.M. was supported by the Ontario Ministry of Health and Long Term Care through the Ontario Neurotrauma Foundation. L.A.B. is a Canada Research Chair in Neurobiology of Motor Learning and a Michael Smith Foundation for Health Research Career Investigator. The authors thank the children and parents who participated in the study, and Alex Rauscher, PhD and Steven Miller, MAS MDCM for their assistance with data processing and interpretation.

References

[1] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th Edition: Text revision. Washington, DC: American Psychiatric Association; 2000.
[2] Querne L, Berquin P, Vernier-Hauvette MP, et al. Dysfunction of the attentional brain network in children with developmental coordination disorder: A fMRI study. Brain Res 2008;1244:89–102.
[3] Kashiwagi M, Iwaki S, Narumi Y, Tamai H, Suzuki S. Parietal dysfunction in developmental coordination disorder: A functional MRI study. Neuroreport 2009;20:1319–24.
[4] Zwicker JG, Missiuna C, Harris SR, Boyd LA. Brain activation of children with developmental coordination disorder is different than peers. Pediatrics 2010;126:e678–86.
[5] Zwicker JG, Missiuna C, Harris SR, Boyd LA. Brain activation associated with motor skill practice in children with developmental coordination disorder: An fMRI study. Int J Dev Neurosci 2011;29:145–52.
[6] Yoshida S, Hayakawa K, Yamamoto A, et al. Quantitative diffusion tensor tractography of the motor and sensory tract in children with cerebral palsy. Dev Med Child Neurol 2010;52:935–40.
[7] Zwicker JG, Missiuna C, Boyd LA. Neural correlates of developmental coordination disorder: A review of hypotheses. J Child Neurol 2009;24:1273–81.
[8] Patestas MA, Gartner LP. A textbook of neuroanatomy. Malden, MA: Blackwell Publishing; 2006. 219–240.
[9] Geuze RH. Postural control in children with developmental coordination disorder. Neural Plast 2005;12:183–96.

[10] Henderson SE, Sudgen DA. Movement Assessment Battery for Children (MABC-2). 2nd ed. London: Harcourt Assessment; 2007.
[11] Wilson BN, Kaplan BJ, Crawford SG, Roberts G. Developmental Coordination Disorder Questionnaire 2007 (DCDQ'07). Available at: http://www.dcdq.ca. Accessed August 6, 2011.
[12] Missiuna C, Pollock N, Egan M, DeLaat D, Gaines R, Soucie H. Enabling occupation through facilitating the diagnosis of developmental coordination disorder. Can J Occup Ther 2008;75:26–34.
[13] Kaufman AS, Kaufman NL. KBIT-2 Kaufman Brief Intelligence Test. 2nd ed. Minneapolis: NCS Pearson; 1997.
[14] Conners CK. Conners' Rating Scales—Revised (CRS-R), technical manual. Toronto: Ontario, Canada: Multi-Health Systems; 2001.
[15] Mori S, Barker PB. Diffusion magnetic resonance imaging: Its principle and applications. Anat Rec 1999;257:102–9.
[16] Jiang H, van Zijl PC, Kim J, Pearlson GD, Mori S. DtiStudio: Resource program for diffusion tensor computation and fiber bundle tracking. Comput Methods Programs Biomed 2006;81:106–16.
[17] Adams E, Chau V, Poskitt KJ, Grunau RE, Synnes A, Miller SP. Tractography-based quantification of corticospinal tract development in premature newborns. J Pediatr 2010;156:882–8.
[18] Schmithorst VJ, Wilke M, Dardzinski BJ, Holland SK. Correlation of white matter diffusivity and anisotropy with age during childhood and adolescence: A cross-sectional diffusion-tensor MR imaging study. Radiology 2002;222:212–8.
[19] Dewey D, Kaplan BJ, Crawford SG, Wilson BN. Developmental coordination disorder: Associated problems in attention, learning, and psychosocial adjustment. Hum Mov Sci 2002;21:905–18.
[20] Trivedi R, Agarwal S, Shah V, et al. Correlation of quantitative sensorimotor tractography with clinical grade of cerebral palsy. Neuroradiology 2010;52:759–65.
[21] Caeyenberghs K, Leemans A, Geurts M, et al. Brain-behavior relationships in young traumatic brain injury patients: DTI metrics are highly correlated with postural control. Hum Brain Mapp 2010;31:992–1002.
[22] Beaulieu C. The basis of anisotropic water diffusion in the nervous system: A technical review. NMR Biomed 2002;15:435–55.
[23] Mukherjee P, Miller JH, Shimony JS, et al. Diffusion-tensor MR imaging of gray and white matter development during normal human brain maturation. AJNR 2002;23:1445–56.
[24] Song SK, Sun SW, Ramsbottom MJ, Chang C, Russell J, Cross AH. Dysmyelination revealed through MRI as increased radial (but unchanged axial) diffusion of water. Neuroimage 2002;17:1429–36.
[25] Barnea-Goraly N, Lotspeich LJ, Reiss AL. Similar white matter aberrations in children with autism and their unaffected siblings: A diffusion tensor imaging study using tract-based spatial statistics. Arch Gen Psychiatry 2010;67:1052–60.
[26] Rollins NK, Vachha B, Srinivasan P, et al. Simple developmental dyslexia in children: Alterations in diffusion-tensor metrics of white matter tracts at 3 T. Radiology 2009;251:882–91.